**QUIZ 19th July 2017 (answers below)**

1. **What is a nasal septal haematoma?**

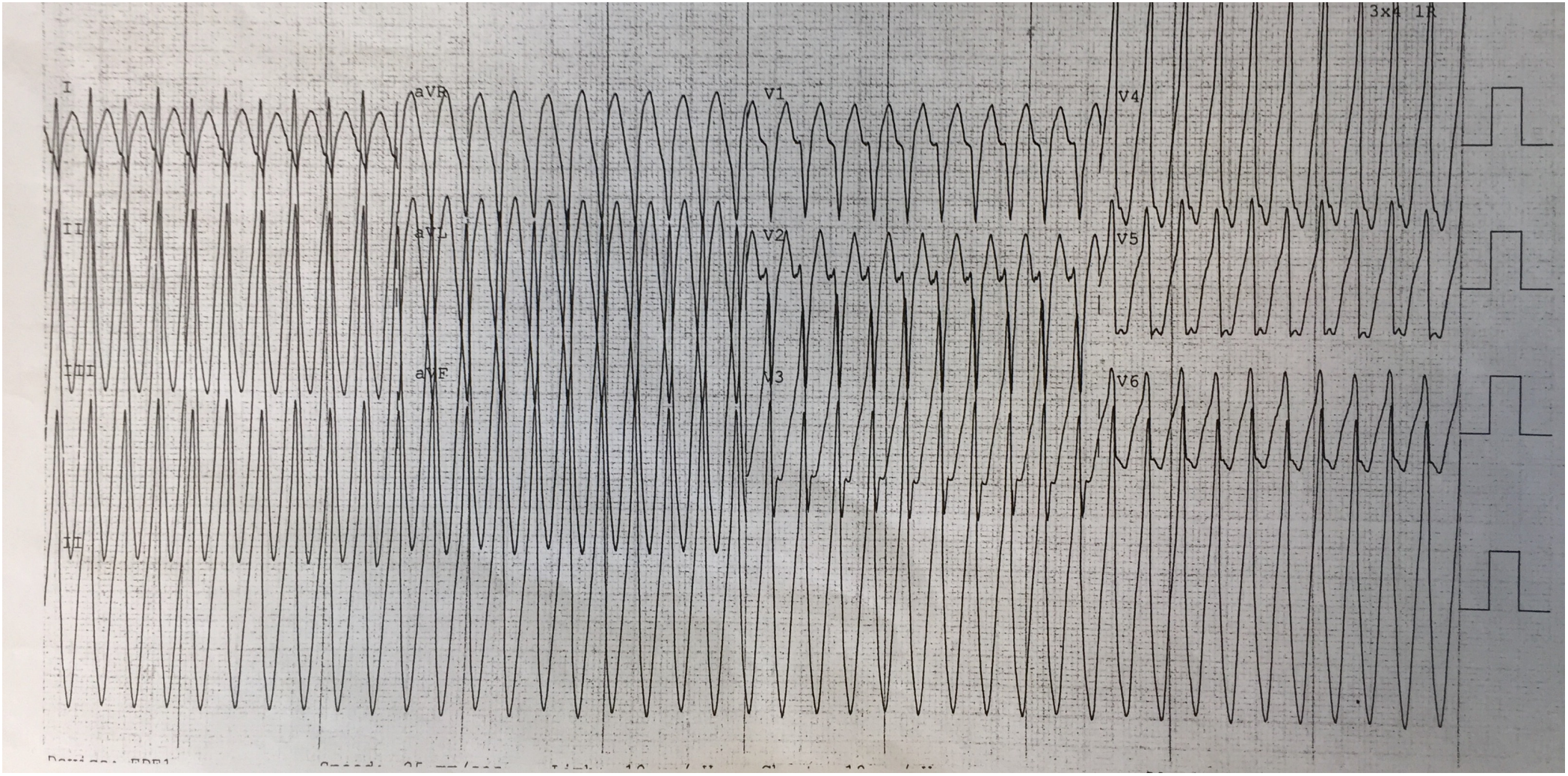
2. **What are the symptoms and signs of local anaesthetic toxicity?**

3. **What is the role of intravenous lipid emulsion in toxicology?**

4. **What is alcohol ketoacidosis?**

5. **Describe and interpret the following ECG.**

28 year old male
Hit in chest while playing football 2 hours prior
Felt chest discomfort, palpitations, SOB and light headed
Self-presented to ED



**QUIZ answers 19th July 2017**

1. **What is a nasal septal haematoma?**

*A nasal septal haematoma is the development of a haematoma between the nasal septal cartilage and the overlying mucoperichondrium. Damage to the septal cartilage can occur within 24 hours and if untreated it can rapidly lead to irreversible septal destruction and substantial nasal deformity requiring extensive reconstruction.*

*It is thought that trauma causes rupture of small submucosal vessels leading to the haematoma. The haematoma then causes pressure related ischaemia of the cartilage and subsequent cartilage necrosis. Destruction of the septum affects the structure of the nose and over time can lead to the classic "saddle" nose.*

*Nasal septal abscess is another complication of the haematoma that requires emergency management as it can lead to orbital cellulitis, meningitis, cavernous sinus thrombosis as well as septal destruction.*

*Nasal septal haematoma can developed following relatively minor trauma to the nose, particularly in children where the cartilage can buckle easily.*

*Nasal septal haematoma is seen as a fluctuant, "cherry red" swelling of the septum that is usually bilateral. Probing with a cotton bud will demonstrate that it is a boggy swelling, in contrast to a deviated septum, which is firm.*

*Management of nasal septal haematoma involves urgent drainage and intravenous antibiotics.*

Sanyaolu L Nasal Septal Haematoma BMJ 2014;349:g6075

2. **What are the symptoms and signs of local anaesthetic toxicity?**

*Early symptoms are usually neurological and include:*

*Dizziness*
*Tinnitus*
*Perioral numbness*
*Anxiety*
*Confusion*

*More severe signs are:*

*CNS – seizures, coma*
*CVS – bradycardia, hypotension, arrhythmias including VF and asystole*
*Respiratory depression, apnoea*

*CNS signs usually develop first, except following massive IV injection where cardiac arrest may be the first sign.*

## 3. What is the role of intravenous lipid emulsion in toxicology?

*Intravenous lipid emulsion (intralipid) has a role in local anaesthetic-induced cardiovascular collapse, resistant to standard resuscitation therapies. It may also have a role in refractory cardiac instability in poisoning with other highly lipid soluble agents (eg. propanolol, tricyclic antidepressants and verapamil).*

*Adult dosing guideline at SVH ED is 100mL bolus followed by 400mL over 20 minutes. Further boluses can be given as needed up to 8mL/kg as a maximum.*

*The optimum dose is not yet established. Little is known about short and long term adverse effects. Use of intralipid outside the above indications is not justified.*

Murray et al Toxicology Handbook 3rd Ed. 2015 Elsevier

## 4. What is alcohol ketoacidosis?

*Alcoholic ketoacidosis is an uncommon acute metabolic acidosis that usually only occurs in people who chronically misuse alcohol and are malnourished. The ketosis is precipitated by an alcohol binge, which is then stopped by the development of abdominal pain and vomiting (eg. from pancreatitis or gastritis). Presentation to hospital is around 48 hours later.*

*The metabolism of ethanol to acetaldehyde and then to acetic acid each converts NAD+ to NADH, resulting in a "redox shift" of the NAD+/NADH ratio towards NADH. This shift suppresses gluconeogenesis and may result in hypoglycaemia.*

*The resulting acetic acid can be used for energy or linked with co enzyme A to form acetyl-CoA, which can go on to ketogenesis. During active alcohol consumption, there is regeneration of the acetic acid, which inhibits peripheral lipolysis, and this limits fatty acid delivery to the liver, preventing significant ketogenesis.*

*When active alcohol consumption stops, there is no further acetic acid to inhibit lipolysis. Abdominal pain and vomiting cause a fasting state and dehydration. As ethanol levels begin to fall, alcohol withdrawal causes an increase in catecholamines and cortisol which in turn amplify the hormonal responses to fasting (low insulin, high glucagon), causing a marked increase in lipolysis and ketogenesis. There is reduced renal clearance of the ketones due to dehydration.*

*The disorder is corrected with fluid resuscitation and dextrose administration. IV thiamine should be given and electrolytes (K, Mg, and PO4) corrected and monitored. Alcohol withdrawal requires benzodiazepines and monitoring. The cause of the abdominal pain and vomiting also needs to be addressed.*

5. **Describe and interpret the following ECG.**

*Regular tachycardia 250/min*
*Wide complexes >0.2 sec*
*= Ventricular tachycardia until proven otherwise*

*Factors against this being ventricular tachycardia*
*No fusion or capture beats*
*No evidence of AV dissociation*
*There is not precordial concordance (V1-2 seems negative and V3-6 positive)*

*The ECG cannot reliably distinguish VT from SVT with aberrancy.*
*The ECG is good at ruling in VT, but not good at ruling out VT*

*There is a bunch of algorithms:*
*Brugada, Bayesian (Vereckei aVR, Griffith, Lead II R-wave-peak-time)*
*All do very poorly in terms of sensitivity when studied*

➔ *Safest to assume all wide complex tachycardias are VT*

*This patient was a 28 year old man who had been hit in the chest while playing football 2 hours prior. He self presented to a regional ED complaining of chest discomfort, palpitations, SOB and light headedness.*

*He received 200J synchronised shock --> Torsades*
*Second shock --> atrial fibrillation*
*IV amiodarone 300mg --> sinus rhythm*
*Commenced metoprolol*

*Investigations*
*CMRI – normal, no evidence of ARVC*
*TTE – normal*
*CTCA – normal*
*EPS study showed normal cardiac conduction times, dual AV nodal pathways and inducible AV nodal tachycardia with aberrancy, no inducible VT. The slow pathway was ablated.*

➔ *So rhythm was SVT with aberrancy but safely and managed as VT*